

# Food-borne botulism in Canada, 1971-84

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Sixty-one outbreaks of food-borne botulism involving a total of 122 cases, of which 21 were fatal, were recorded from 1971 to 1984 in Canada. Most occurred in northern Quebec, the Northwest Territories or British Columbia. Of the 122 victims 113 were native people, mostly Inuit. Most of the outbreaks (59%) were caused by raw, parboiled or "fermented" meats from marine mammals; fermented salmon eggs or fish accounted for 23% of the outbreaks. Three outbreaks were attributed to home-preserved foods, and one outbreak was attributed to a commercial product. The causative *Clostridium botulinum* type was determined in 58 of the outbreaks: the predominant type was E (in 52 outbreaks), followed by B (in 4) and A (in 2). Renewed educational efforts combined with a comprehensive immunization program would significantly improve the control of botulism in high-risk populations.

De 1971 à 1984 on enregistre au Canada 61 faits de botulisme d'origine alimentaire répartis en 122 cas dont 21 mortels. Ces faits se produisent surtout dans le nord du Québec, aux Territoires du Nord-Ouest et en Colombie-britannique. Des 122 malades, 113 sont des aborigènes, surtout des Inuit. La plupart des infections (59%) proviennent de la viande de mammifères marins qui a été consommée crue, ébouillantée ou après "fermentation"; dans 23% il s'agit d'oeufs de saumon fermentés ou de chair de poisson, trois fois de conserves domestiques et une fois d'un produit du commerce. Dans

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# Clinical and Community Studies

58 faits on connaît le type du *Clostridium botulinum* causal: E (52 faits), B (4) et A (2). La prévention du botulisme au sein des populations à risque élevé comprend la dissémination des connaissances et un vaste programme de vaccination.

**B**otulism outbreaks have been recorded in Canada since 1919 and have been reviewed to the end of 1973.<sup>1-3</sup> Until the early 1950s nearly all the documented outbreaks were caused by home-canned foods, and most of the victims were of European descent. From then on, the peccant foods were mainly unprocessed meats from marine mammals or "fermented" salmon eggs, and most of the victims were native people of northern Canada or the coastal regions of British Columbia. In this paper we review the botulism outbreaks that have been documented in Canada since 1973. Since the published summaries for the preceding years<sup>3,4</sup> were somewhat incomplete, we have also included the period 1971-73 in this review.

## Features of outbreaks

Sixty-one outbreaks of food-borne botulism involving a total of 122 cases, 21 of which were fatal, were confirmed in the period 1971-84 (Table I). During this time only a single case of infant botulism was confirmed,<sup>5</sup> and no cases of wound botulism were recorded. Nearly 70% of the outbreaks (41) occurred in the period May to October, with the peak in July (11). The month with the lowest number of outbreaks (one) was January.

## Geographic distribution

The main focus of the outbreaks was in regions of northern Quebec and the Northwest

Table 1—Outbreaks of food-borne botulism in Canada, 1971–84

Year	Location	Suspected food	No. of cases	No. of deaths	Toxin type
1971	Terrace, BC	Salmon eggs	2	0	E
	Fort Chimo, PQ	Beluga meat	9	3	E
	Fort Chimo, PQ	Seal meat	1	0	E
1972	Mississauga, Ont.	Home-salted pork	1	0	B
	Fort Chimo, PQ	Caribou meat	2	0	?
	Bella Bella, BC	Salmon eggs	6	0	E
1973	Bella Bella, BC	Salmon eggs	1	0	E
	Payne Bay, PQ	Beluga meat	3	1	E
	Fort Chimo, PQ	Seal meat	1	1	E
1974	Montreal, PQ	Commercial bottled mushrooms	1	0	B
	Fort Chimo, PQ	Seal meat	1	0	E
	Cambridge Bay, NWT	Caribou meat	2	1	E
1975	Payne Bay, PQ	Walrus meat	2	0	E
	Frobisher Bay, NWT	Arctic char	3	1	E
	North West River, Nfld.	Seal meat	3	1	E
1976	Hartley Bay, BC	Salmon eggs	2	2	E
	Port Burwell, NWT	Seal flippers	5	0	E
	Port Burwell, NWT	Unknown	1	0	E
1977	Fort Chimo, PQ	Seal meat	1	0	E
	Fort Chimo, PQ	Unknown	1	0	E
	Frobisher Bay, NWT	Seal meat	2	0	?
1978	Fort Chimo, PQ	Seal meat	1	0	E
	Fort Chimo, PQ	Arctic char	4	0	E
	Fort Chimo, PQ	Whale meat	10	1	E
1979	Baie-aux-Feuilles, PQ	Whale meat	1	1	E
	Fort Chimo, PQ	Caribou and/or seal meat	1	1	E
	Happy Valley, Nfld.	Seal meat	1	1	E
1980	Saglouc, PQ	Whale meat	3	0	E
	Lake Harbour, NWT	Seal meat	2	1	E
	Coppermine, NWT	Dried fish	1	0	E
1981	Fort Chimo, PQ	Seal meat	1	1	E
	Kitimat, BC	Salmon eggs	1	0	E
	Payne Bay, PQ	Walrus meat	3	0	E
1982	Cape Dorset, NWT	Walrus meat	2	0	E
	Fort Chimo, PQ	Unknown	2	0	E
	Fort Chimo, PQ	Seal meat	1	0	E
1983	Klemtu, BC	Salmon eggs	1	0	E
	Fort Chimo, PQ	Seal meat	2	0	E
	Fort Chimo, PQ	Seal meat	3	1	E
1984	Fort Chimo, PQ	Caribou meat	1	0	E
	Fort Chimo, PQ	Seal meat	1	0	E
	Poste-de-la-Baleine, PQ	Walrus meat	3	1	?
1985	Tuktoyaktuk, NWT	<i>Muktuk</i> *	1	0	E
	Fort Chimo, PQ	Seal meat	1	0	E
	Fort Chimo, PQ	Seal meat	2	0	E
1986	Fort Chimo, PQ	Seal meat	2	0	E
	Coppermine, NWT	<i>Urraq†</i>	2	0	E
	Vancouver, BC	Salmon eggs	2	0	E
1987	Queen Charlotte City, BC	Home-smoked salmon	2	0	E
	Fort Chimo, PQ	Seal meat	1	0	E
	Fort Chimo, PQ	Seal meat	1	0	E
1988	Surrey, BC	Home-canned corn	1	0	A
	North Vancouver, BC	Home-smoked salmon	1	1	E
	North Vancouver, BC	Home-smoked salmon	2	0	B
1989	Payne Bay, PQ	Seal meat	1	0	E
	Payne Bay, PQ	Seal meat	2	0	E
	St. Anthony, Nfld.	Seal meat	1	1	E
1990	Baker Lake, NWT	Lake trout	1	1	B
	Coppermine, NWT	Seal meat	1	0	E
	Inuvik, NWT	<i>Muktuk</i>	2	1	E
1991	North Vancouver, BC	Home-canned vegetables	1	0	A
	Inuvik, NWT	<i>Muktuk</i>	1	0	E
Total			122	21	

\*Chunks of skin with blubber and meat of the white whale (beluga).

†Uncooked seal flippers in seal oil.

Territories adjacent to Ungava Bay and Hudson Strait. The remaining outbreaks occurred mainly around Inuvik and Coppermine, NWT, and in the coastal regions of British Columbia.

The number of recorded outbreaks in Quebec and the Northwest Territories represents a considerable increase over the preceding 20-year period (Table II). The increase may indicate a higher frequency of outbreaks or may merely reflect a greater awareness of botulism in these areas. On the other hand, the frequency of documented botulism declined in British Columbia and Newfoundland. The decrease in British Columbia may be attributable to the efforts of public health agencies to discourage the fermentation of salmon eggs,<sup>4</sup> which were responsible for 18 of the 26 outbreaks in 1951-70 and for 7 of the 12 in 1971-84. The pattern in Newfoundland is difficult to assess; most of the outbreaks reported from this province (11 of 14) occurred in the short period 1956-62.

### Peccant foods

Nearly 60% of the outbreaks (36) were caused by meats from marine mammals, mostly seals, that had been eaten raw or undercooked or had undergone a faulty fermentation process (Table III).

Table II—Geographic distribution of botulism outbreaks, 1919-84

Province or territory	No. of reported outbreaks; period		
	1919-50	1951-70	1971-84
Yukon Territory and Northwest Territories	3	6	14
British Columbia	3	26	12
Quebec	0	3	31
Newfoundland	0	11	3
Other	4	1	1
Total	10	47	61

Outbreaks caused by raw or undercooked meats can generally be linked to the habit of keeping meats at ambient temperatures for some time. For example, in the outbreak in North West River, Nfld., in 1974 the meat had been kept hanging in the kitchen to dry for nearly 2 weeks; in one of the outbreaks in Port Burwell, NWT, in 1975 the seal flippers had been stored on the roof; and the whale meat implicated in the outbreak in Sagloug, PQ, in 1976 had been deliberately held for a week to decompose for a desired flavour and texture. Each of these conditions would support growth of the nonproteolytic, psychrotolerant forms of *Clostridium botulinum*, which grow at temperatures as low as 4°C. The contaminating spores may come from the animals' entrails as well as from the environment; spores of nonproteolytic *C. botulinum*, particularly type E, are abundant in the coastal areas and around inland lakes of the more frigid regions of the northern hemisphere (i.e., Alaska, Canada, Scandinavia, the USSR, northern Iran and Hokkaido, Japan<sup>6</sup>).

Caribou meat was implicated in three of the recorded outbreaks. However, in contrast to sea mammals, caribou are unlikely to carry *C. botulinum* spores. According to the survey data of one of us (L.G.),<sup>7</sup> caribou droppings are essentially free of the organism. Whenever caribou meat is incriminated in an outbreak, the meat has likely been contaminated through contact with soil or with meat or oil from sea mammals.

Fermented meats responsible for outbreaks were *urraq* (uncooked seal flippers in seal oil) and *muktuk* (chunks of skin with blubber and meat of the white whale [beluga]).<sup>2,8,9</sup> These foods contain few fermentable carbohydrates and therefore do not undergo the rapid reductions in pH that occur in established fermentation processes for meats and vegetables. As a result, *C. botulinum* rapidly grows.

Seven outbreaks were caused by fermented salmon eggs, which are prepared by means of similarly unsafe procedures. The eggs are generally fermented whole in their own juice, with or

Table III—Foods and ethnic groups involved in botulism outbreaks, 1971-84

Food	No. of outbreaks	No. of cases	No. of deaths	Ethnic group	Toxin type (and no. of outbreaks)
Meat from marine mammals					
Raw or parboiled	32	73	13	Inuit	E
Fermented	4	6	1	Inuit	E
Caribou meat, raw or parboiled	3	5	1	Inuit	E
Fish					
Raw, parboiled or dried	4	9	2	Inuit	E (3), B (1)
Smoked	3	5	1	White	E (2), B (1)
Salmon eggs, fermented	7	15	2	Amerindian	E
Preserved meats or vegetables					
Home-preserved	3	3	0	White	A (2), B (1)
Commercial	1	1	0	White	B
Unknown	4	5	1	Inuit	E

without added salt; they may also be kneaded into a firm mass before curing.<sup>2,10</sup> Lack of carbohydrate precludes adequate acid fermentation, and the food becomes putrid instead. In a survey of 26 lots of freshly fermented salmon eggs the mean pH was 5.9 (unpublished data). However, the pH of a few lots of fermented eggs of chum salmon (*Oncorhynchus keta*) ranged from 4.2 to 5.1, which would inhibit or delay growth of type E and other nonproteolytic forms of *C. botulinum*.

Three outbreaks, all in 1983, were caused by home-smoked salmon. In each case the salmon had been held too long and with little heat in the smoke chamber or had been kept for some time at ambient temperatures after smoking,<sup>11</sup> or both.

Only three outbreaks caused by home-preserved meat or vegetables (salted ham in one and vegetables preserved in Mason jars in two) were recorded. Contrary to established guidelines,<sup>12</sup> at least one of the jars of vegetables had been processed in an open water bath and consumed without being reheated.<sup>11</sup> The small number of home-canned foods incriminated in Canada is in sharp contrast to the numbers reported in the United States,<sup>13</sup> where in the period 1971-77 nearly half of the botulism outbreaks (46 of 102) were caused by home-canned foods, mostly vegetables.

A single commercial product, bottled marinated mushrooms imported from the United States, was incriminated in an outbreak, in Montreal in 1973; inadequate acidification was likely the cause of toxin production.<sup>14</sup>

Table III shows distinct links between the kind of food involved and both the victims' ethnic group and the causative *C. botulinum* type. Raw, parboiled, dried or fermented meats and fish affected only Inuit, fermented salmon eggs caused outbreaks only among Amerindians, and smoked fish and preserved meats or vegetables affected only people of European descent. Inuit were involved in 77% of the outbreaks (47) and accounted for 80% of all cases (98) and 86% of the fatal cases (18).

At least three of the four outbreaks caused by preserved meats or vegetables involved proteolytic forms of the organism. All the other outbreaks, including the two due to type B caused by fish, involved only nonproteolytic *C. botulinum*.

#### Fatality rates

The overall death rate during the period studied was 17%, compared with 50% to 60% a few decades earlier (Table IV). The most significant decrease occurred in the period 1966-75. The lower rates are attributable primarily to improved general health services and life support techniques in remote regions, the use of antitoxin, which was first made commercially available in 1964 (by Connaught Laboratories, Toronto), and better services for transporting patients to medical centres. For example, in the outbreak in Cape Dorset,

NWT, in 1978 the initial diagnosis of botulism was made by the resident nurse, who had a supply of antiserum, and arrangements were made within hours for flying the patients to Frobisher Bay, NWT. The two patients involved had serum toxin levels of 40 and 100 mouse lethal doses per millilitre and would almost certainly have died without this effective service.<sup>19</sup> Another factor in the declining fatality rates in recent years may be an increase in the number of nonfatal cases recognized and recorded. The distribution of incriminated *C. botulinum* types does not seem to be a factor, as type E has been the predominant type since before the rates decreased.

Among the outbreaks summarized in Table III are four not listed in Dolman's review;<sup>3</sup> details are given in the footnote.

#### Laboratory confirmation

The diagnosis of botulism is considered confirmed when, in addition to the characteristic clinical syndrome, botulinal toxin or viable *C. botulinum* is shown in clinical specimens or the

Table IV—Fatality rates of botulism cases, 1919-84

Period	No. of outbreaks	No. of cases	No. of deaths	Fatality rate, %
1919-50	10	57	32	56
1951-60	15	33	19	58
1961-65	21*	41	21	51
1966-70	11	25	9	36
1971-75	23	55	10	18
1976-80	18	39	7	18
1981-84	20	28	4	14

\*Includes four outbreaks not reported in Dolman's review:<sup>3</sup> one of type A due to home-canned asparagus in Grand Forks, BC, in 1961 (two cases, one of which was fatal)<sup>15</sup> (E.J. Bowmer: personal communication); one of type E due to salmon eggs in Bella Bella, BC, in 1964 (one case);<sup>16</sup> one of type E due to salmon eggs in Queen Charlotte City, BC, in 1964 (two fatal cases);<sup>17</sup> and one of type A due to home-canned chicken in Halifax in 1965 (one case).<sup>18</sup>

Table V—Results of analysis of clinical specimens and food for botulinal toxin or *Clostridium botulinum*

Specimen	Analysis	No. of specimens analysed	No. (and %) of specimens with positive results
Serum	Toxin	52	37 (71)
Gastric contents	Toxin	35	5 (14)
	<i>C. botulinum</i>	26	14 (54)
Stool	Toxin	18	7 (39)
	<i>C. botulinum</i>	14	13 (93)
Liver	Toxin	1	0
	<i>C. botulinum</i>	1	1
Food	Toxin	28	20 (71)
	<i>C. botulinum</i>	32	24 (75)

suspected food.<sup>20</sup> Of the various analyses, the detection of toxin in the serum provided the largest number of laboratory confirmations (37) (Table V). Occasionally we failed to detect toxin in the serum of severely ill patients, while serum from patients with milder cases was highly toxic.<sup>21</sup> The most critical factors that determine toxin levels are likely the time of sampling and the degree of toxin binding at the myoneural receptor site.

Demonstration of viable *C. botulinum* or toxin in the suspected food provided the next two largest numbers of confirmations (24 and 20 respectively). A higher proportion of positive results (93%) was obtained in analysis of stool specimens for *C. botulinum*, but the number of such analyses was relatively small because stools frequently are not included among the specimens submitted and because culturing of stools may be forgone or discontinued if another toxin test gives positive results.

In one fatal case *C. botulinum* was isolated from the liver.<sup>22</sup> Liver specimens generally are not examined for the organism but have occasionally been shown to contain botulinum toxin.<sup>23</sup>

Detection rates of 33% to 34% for toxin in both serum and stools and of 60% for *C. botulinum* in stools have been reported in patients with botulism in the United States.<sup>24</sup> The higher rate in serum in Canadian outbreaks, 71%, may be due to the predominant involvement of type E in this country. In France, where most outbreaks are also caused by nonproteolytic forms of the organism (though of type B), the detection rate has been reported to be essentially the same as that in Canada.<sup>25</sup>

### Protection of high-risk populations

It is apparent that the Inuit are by far at highest risk for botulism (30 cases per 100 000 population annually, on the basis of documented cases). Stuart and colleagues<sup>26</sup> estimated that the Inuit of Alaska and Canada suffered botulism 500 times more frequently than did the remaining population of North America. Given that 98 of the 122 cases recorded in 1971-84 were in Inuit and that Inuit constitute only 0.1% of the population of Canada, the ratio for Canadian Inuit (4000:1) is nearly 10 times higher than the estimate of Stuart and colleagues. The true ratio may be several times higher still because a significant proportion of cases in the north is likely to remain undetected. Also, not included in our summary are suspected cases of botulism that could not be confirmed by laboratory analysis: because of transportation problems, clinical specimens and food samples had often deteriorated to the extent that any toxin present would likely have broken down.

Two measures have repeatedly been proposed to lessen the botulism hazards in the north: better public health education<sup>4</sup> and improved medical services.<sup>10</sup> The latter measure has had a marked

effect, as evidenced by the reduction in fatality rates from over 50% to less than 20%. Educational efforts may have had an effect in reducing the incidence of botulism due to fermented salmon eggs in British Columbia, but we have no evidence of comparable success in Inuit communities.

A significant factor in the improved treatment of botulism appears to be the early administration of trivalent antitoxin A,B,E.<sup>27,28</sup> In most outbreaks all patients with any sign of botulism receive a dose of antitoxin, which usually results in the prompt disappearance of the symptoms, even in patients with some respiratory impairment. However, antitoxin loses its neutralizing effect as the toxin becomes fixed on the nerve terminals.<sup>29</sup> This loss in efficacy is well exemplified by the fact that in some cases the patient did not recover despite administration of large doses of antitoxin and good supportive care.

In our experience, Inuit have not shown any hypersensitivity to horse serum antitoxin. However, because of their deep-rooted eating habits, they are prone to re intoxication, despite their awareness and fear of botulism. Two of the cases in 1971-84, one of them fatal, were in Inuit who had survived a previous outbreak of botulism. This raises questions about the value of prophylactic administration of antitoxin to symptomless members of a group that shared the suspected food. Prophylaxis should be applied with circumspection in such cases because it might jeopardize treatment of a subsequent and more severe intoxication.

Stuart and colleagues<sup>26</sup> recommended immunization of the Inuit population with toxoid. Toxoid immunization was also considered in Alaska.<sup>30</sup> Since 1958 laboratory personnel world wide who handle *C. botulinum* have been immunized with pentavalent (A-E) toxoid that is listed as an "experimental drug".<sup>31</sup> Though the toxoid is immunogenically effective,<sup>32</sup> cases of severe local reactions seemed to preclude its more extended use. However, such reactions could be eliminated by increasing the intervals between administration of booster doses and could be reduced by detoxification with reduced formaldehyde concentrations.<sup>32</sup> Furthermore, toxoids for Inuit could be simplified by including only type E or types E and B in the product.

There is no doubt that renewed educational efforts combined with a comprehensive immunization program would significantly improve the control of botulism in the Inuit.

### References

1. Dolman CE: Additional botulism episodes in Canada. *Can Med Assoc J* 1954; 71: 245-249
2. Idem: Further outbreaks of botulism in Canada. *Can Med Assoc J* 1961; 84: 191-200
3. Idem: Human botulism in Canada (1919-1973). *Can Med Assoc J* 1974; 110: 191-197, 200
4. Bowmer EJ, Wilkinson DA: Botulism in Canada, 1971-74 [C]. *Can Med Assoc J* 1976; 115: 1084, 1086

5. McCurdy DM, Krishnan C, Hauschild AHW: Infant botulism in Canada. *Can Med Assoc J* 1981; 125: 741-743
6. Hauschild AHW: Microbial problems in food safety with particular reference to *Clostridium botulinum*. In Graham HD (ed): *The Safety of Foods*, 2nd ed, Avi, Westport, Conn, 1980: 68-107
7. Botulism in Canada — caribou meat as a source of intoxication? *Can Dis Wkly Rep* 1985; 11: 93-94
8. Dolman CE: Botulism as a world health problem. In Lewis KH, Cassel K (eds): *Botulism* (PHS publ no 999-FP-1), Robert A. Taft Sanitary Engineering Center, Cincinnati, Ohio, 1964: 5-32
9. Botulism — Northwest Territories. *Can Dis Wkly Rep* 1983; 9: 55-56
10. Dolman CE, Iida H: Type E botulism: its epidemiology, prevention and specific treatment. *Can J Public Health* 1963; 54: 293-308
11. Botulism in Canada — summary for 1983. *Can Dis Wkly Rep* 1984; 10: 85-87
12. *Canning Canadian Fruits and Vegetables* (publ no 1560E), Agriculture Canada, Communications Branch, Ottawa, 1982
13. *Botulism in the United States, 1899-1977. Handbook for Epidemiologists, Clinicians and Laboratory Workers*, Centers for Disease Control, Atlanta, 1979
14. Todd E, Chang PC, Hauschild A et al: Botulism from marinated mushrooms. *Proc IV Int Congr Food Sci Technol* 1974; 3: 182-188
15. Meyer KF, Eddie B: *Sixty-five Years of Human Botulism in the United States and Canada*, George Williams Hooper Foundation, U of Calif, San Francisco Medical Center, San Francisco, 1965: 20
16. Moore PE: Botulism — Northwest Territories. *Epidemiol Bull* 1964; 8: 77
17. Epidemiological bulletin. *Can Med Assoc J* 1964; 90: 48
18. Van Rooyen CE, Snell E: Botulism — Nova Scotia. *Epidemiol Bull* 1965; 9: 97
19. Botulism at Cape Dorset, N.W.T. *Can Dis Wkly Rep* 1978; 4: 77-79
20. *Foodborne Disease Outbreaks, Annual Summary 1978*, Centers for Disease Control, Atlanta, 1981
21. Botulism in Canada — summary for 1978. *Can Dis Wkly Rep* 1979; 5: 89-90
22. Botulism in Canada: summary for 1984. *Can Med Assoc J* 1985; 132: 1402
23. Gilbert RJ, Roberts D, Smith G: Food-borne diseases and botulism. In Smith GR (vol ed): *Bacterial Diseases*, vol 3 of Wilson G, Miles A, Parker MT (gen eds): *Topley and Wilson's Principles of Bacteriology, Virology and Immunology*, 7th ed, Williams & Wilkins, Baltimore, 1984: 477-514
24. Dowell VR, McCroskey LM, Hatheway CL et al: Coproexamination for botulinum toxin and *Clostridium botulinum*. *JAMA* 1977; 238: 1829-1832
25. Sebald M, Saimot G: Toxémie botulique. Intérêt de sa mise en évidence dans le diagnostic du botulisme humain de type B. *Ann Microbiol (Paris)* 1973; 124A: 61-69
26. Stuart PF, Wiebe EJ, McElroy R et al: Botulism among Cape Dorset Eskimos and suspected botulism at Frobisher Bay and Wakeham Bay. *Can J Public Health* 1970; 61: 509-517
27. Morris JG: Current trends in therapy of botulism in the United States. In Lewis GE (ed): *Biomedical Aspects of Botulism*, Acad Pr, New York, 1981: 317-326
28. Tacket CA, Shandera WX, Mann JM et al: Equine antitoxin use and other factors that predict outcome in type A foodborne botulism. *Am J Med* 1984; 76: 794-798
29. Smith LDS: *Botulism. The Organism, its Toxins, the Disease*, CC Thomas, Springfield, Ill, 1977: 183
30. Heyward WL, Bender TR: Botulism in Alaska, 1947-1980. In Lewis GE (ed): *Biomedical Aspects of Botulism*, Acad Pr, New York, 1981: 285-289
31. Hardegree MC: Bacterial toxoids: perspectives for the future. *Ibid*: 217-231
32. Anderson JH, Lewis GE: Clinical evaluation of botulinum toxoids. *Ibid*: 233-246

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17. Judson FN, Miller KG, Schaffnit TR: Screening for gonorrhea and syphilis in the gay baths — Denver, Colorado. *Am J Public Health* 1977; 67: 740-742
18. Wolf FC, Judson FN: Intensive screening for gonorrhea, syphilis and hepatitis B in a gay bathhouse does not lower the prevalence of infection. *Sex Transm Dis* 1980; 7: 49-52
19. Thin RN: Health advisers (contact tracers) in sexually transmitted disease. *Br J Vener Dis* 1984; 60: 269-272
20. Judson FN, Wolf FC: Tracing and treating contacts of gonorrhea patients in a clinic for sexually transmitted diseases. *Public Health Rep* 1978; 93: 460-463
21. Potterat JJ, King RD: A new approach to gonorrhea control. *JAMA* 1981; 245: 578-580
22. Potterat JJ, Rothenberg R: The case-finding effectiveness of a self-referral system for gonorrhea: a preliminary report. *Am J Public Health* 1977; 67: 174-176
23. LaBocchetta AC: Comment on control of STD. *Am J Public Health* 1981; 71: 538-539
24. Starcher ET, Kramer MA, Carlota-Orduna B et al: Establishing efficient interview periods for gonorrhea patients. *Am J Public Health* 1983; 73: 1381-1384

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### April

Apr. 27-29, 1986

Fundamental Problems in Breast Cancer  
Banff Springs Hotel  
Secretary, Breast Unit, Cross Cancer Institute, 11560  
University Ave., Edmonton, Alta. T6G 1Z2

### May

May 15-18, 1986

Third Annual Congress of the Canadian Association for  
Medical Anthropology  
Westin Hotel and University of Alberta, Edmonton  
Dr. Janice Morse, Faculty of Nursing, Clinical Sciences  
Bldg., University of Alberta, Edmonton, Alta.  
T6G 2G3; (403) 432-6250

May 28-30, 1986

Suicide: Identifying, Understanding and Helping People  
at Risk  
King's College, London  
Dr. John D. Morgan, Principal, King's College, 266  
Epsworth Ave., London, Ont. N6A 2M3; (519) 433-3491

May 28-30, 1986

The S.M. Dinsdale International Conference in Rehabili-  
tation: "Towards the 21st Century"  
Royal Ottawa Regional Rehabilitation Centre  
Ms. Carol Anne Clarke, Public Relations Officer, Royal  
Ottawa Hospital, 1145 Carling Ave., Ottawa, Ont.  
K1Z 7K4; (613) 722-6521